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Occupational Health in Ontario

Volume 5, Number 1, January 1984

Confidentiality and the Right to Know

Equilibrium of CO Between the Pregnant Mother and the Foetus

Frost Injuries of the Extremities

County Level Standardized Mortality Ratios in Ontario, 1954-1978

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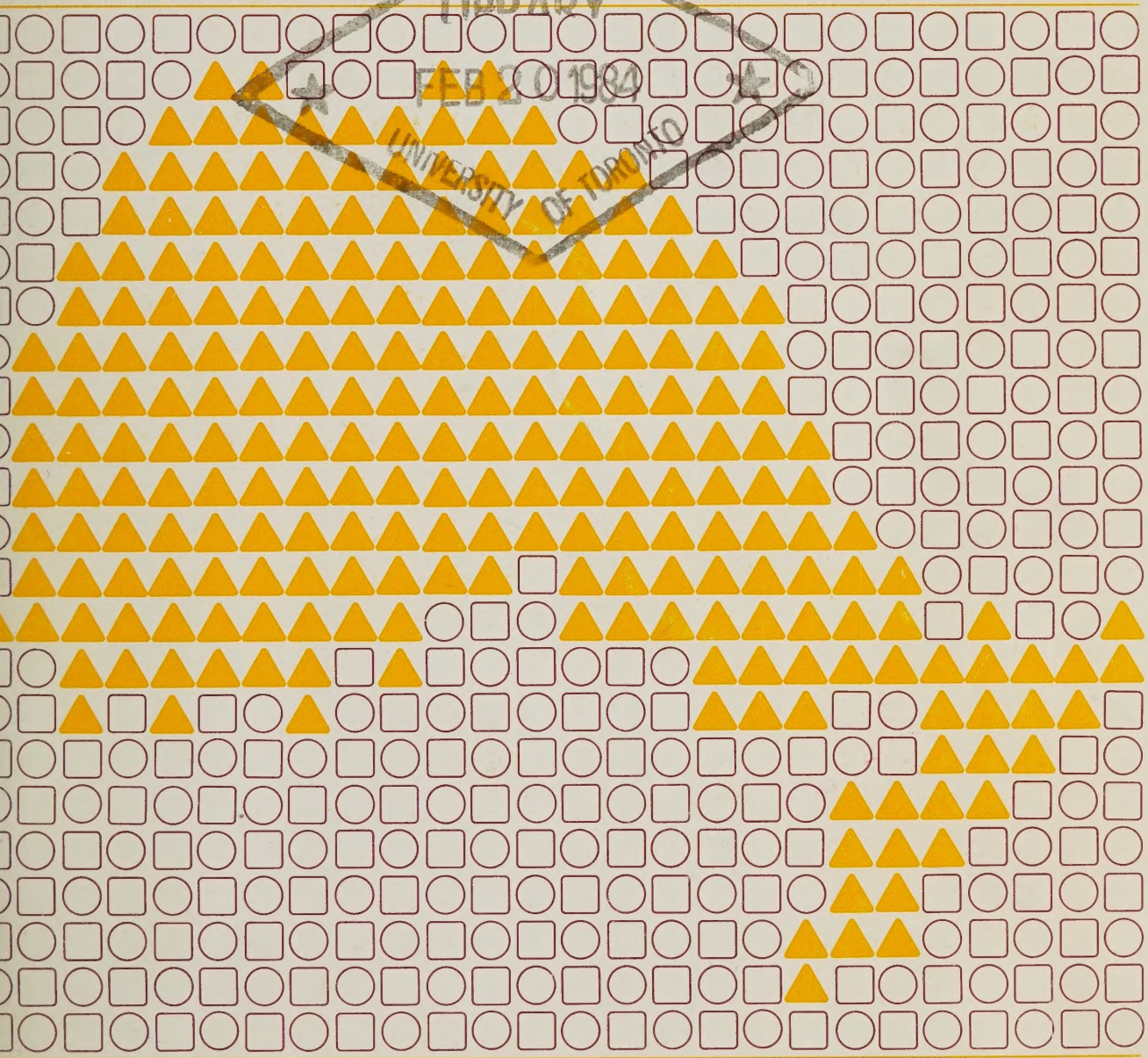

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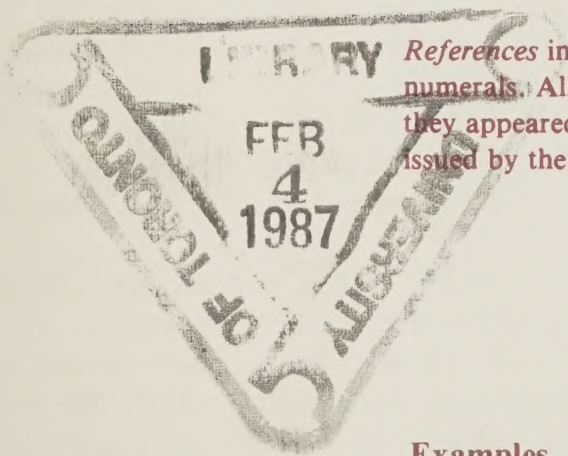

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James R. Nethercott, M.D., F.R.C.P.(C)

Confidentiality and the Right to Know*

Summary

The responsibility of doctors and nurses to make decisions that may affect a worker's employment and employability is discussed. The conflict between the patient's right to confidentiality and the need of a third party for information about the patient's health is explored.

James R. Nethercott, M.D., F.R.C.P.(C), Director, Occupational and Environmental Health Unit, University of Toronto, and the Occupational Health Clinic, St. Michael's Hospital, Toronto, Ontario M5B 1W8

The relationship between worker health and occupation has altered dramatically in the past 200 years. Children no longer succumb to occupationally induced lung disease before reaching adulthood as they did in Dickens' time, for example. Without a doubt, workers today still face real hazards to their health from accidents and work-related diseases. Lung cancer deaths among northern Ontario uranium miners remain a grim reminder to all of us of the continuing threat of occupational disease, even though such disease has been recognized for centuries to be associated with specific workplace conditions.

Legitimate concerns regarding the effects of workplace conditions on health lead workers, government and management to pay progressively more attention to these risks. Successes in this arena, such as the identification of asbestos, cotton dust and radon gas as causes of work-related disease and the correction of such exposures owe much to the insight and persistence of workers and their organizations.

These efforts, which have benefited all workers, not only those who are organized, speak for themselves as clear proof of the importance that labour organizations, such as the Ontario Federation of Labour, place on the protection and promotion of health of Canadian workers. Your organization, and each of you, I know, is committed to the principle that the health of workers is not for sale.

Doctors and nurses have chosen to enter the workplace in increasing numbers in this century. They have, in many instances, given up other professional pursuits to commit their energies entirely to helping make the workplaces of men and women safer. In the past decade I have repeatedly been impressed with the dedication of the professionals whom I have met in the field of occupational health, a dedication which is at least as intense as that of professionals in other fields of medicine and

*Speech given for the Ontario Federation of Labour, Queen's University, Kingston, Ontario, August 16, 1983

nursing. I believe that they are sincere in their desire to make the workplaces to which they devote their time, safe, as do all of us here today.

In this quest for a safe workplace they are often placed in an unavoidably difficult position. Their professional training has led them into situations where decisive action must be taken. A decision about whether to give a dangerous, but life-saving, drug is often very difficult. But we, as patients, expect the doctor to help us understand the situation and counsel us as to the best course of action to be taken. Consider the nurse who, faced with a patient who is bleeding to death, may have to apply a tourniquet to a leg to save the person's life. At the same time, the nurse is taking the risk that this action may cost the person their leg. A terrible decision to have to make in an emergency, yet, clearly, one that is vital. People in these two professions must also live with those situations where they have made carefully weighed decisions which may have adverse effects. They are no less human than any of us.

Increasingly, in the workplace, doctors and nurses are expected to make decisions which may be no less important in the eyes of those affected than the situations I have mentioned. On reflection, I hope that all of us can agree that certain decisions must be made to protect workers from injury or disease that might result from workplace exposure. Someone must be able to examine the worker, understand the work and be able to provide a realistic assessment as to whether the worker is at risk of damage to his or her health if placed in a given job. Someone must accept the ultimate decision-making responsibility.

For example, the rest of us expect that the man driving a school bus, a fork-lift truck or a police car will be able to see well enough that we or our loved ones will not be injured as a result of a driving error. Clearly, then, to protect fellow workers, the rest of the community and the worker in question, a decision must be made about who can enter certain jobs.

These decisions have fallen on nurses and doctors. We are not always likely to agree with all the decisions made by occupational health professionals any more than we are likely to agree with decisions made in their more familiar roles in hospitals and clinics. But we must agree that someone has to make these decisions and that, for the present, they are likely the best professionals to make them.

In what circumstances is a worker likely to meet the doctor or nurse? Three common situations come to mind: The pre-employment evaluation, during medical surveillance programs and when someone has been injured or becomes ill.

Let's consider each of these, beginning with the pre-employment examination. The person seeking a job wants to support himself and his family. Professional assessment is essential to establish that there is no incapacity which would make it hard for the person to do the job or that there is no condition which would be aggravated by the type of work done. Further, it is important to establish that there is no disability that might reasonably lead the doctor to consider that the person might pose a threat to others in the workplace. The worker is often asked to give consent to obtain further medical information from doctors and hospitals. The doctor cannot assess the person properly without the worker's co-operation. The worker knows that any suggestion of illness is unlikely to help in getting the job. Clearly, then, we have a dilemma. But within the limits placed on the doctor or nurse an assessment *is* possible. Consent forms should be completed after the examination and when the worker has had a complete explanation as to what information will be sought, why and from whom. Limiting the scope of such access to one's medical information, and to what is essential for assessment of the ability to do a specific job, is the best course for the worker and the examiner.

Another common interaction with the doctor or nurse is in the medical surveillance programs which are developing increasingly in industry. Periodic examinations are helpful in ensuring that work factors are not hampering worker health. It may be possible to detect unexpected disease early, which would allow rapid intervention and further protection of other workers who may be exposed to the same risks. Laboratory tests may extend the doctor's ability to evaluate and protect the health of the worker. Each procedure or examination needs a separate informed consent, unless it is for a designated substance covered by governmental regulations. The individual patient has the right to know what is found in this examination and to an explanation as to why such an examination is taking place. Usually, there is the right to refuse such an examination unless it is prescribed by law, as in the case of certain designated substances. As such programs are intended to protect and promote health, I would urge workers to participate if the conditions outlined above are met.

Development of a work-related injury or illness will often lead the worker to the nurse or physician in the workplace. Workers who become sick at work or who are injured often receive treatment in the workplace. An examination may take place after an illness or injury to ensure that the worker is well enough to return to work. Injured workers may also have occasion to be examined by doctors employed by third parties, such as insurance companies, the compensation board or an occupational health clinic. This is clearly a decisive type of interaction with the doctor where income for one's self and one's family rests on the decision that is made. The worker, understandably, will approach such encounters with caution. He or she should ensure that the release forms signed are specific to the problem for which the assessment is taking place, and it is important to remember that, in Ontario, the Workers' Compensation Board does not need a consent form to obtain medical information. It is not unreasonable to ask what information is being transmitted by your doctor to another or to the compensation board to ensure that the facts that you have provided are correctly rendered, and to be sure that you understand the interpretation which has been placed on the information by your doctor or doctors.

We all have the right to know what is in our medical files; we have the right to information which is of a factual nature, such as laboratory test results, and to have the doctor's opinion explained by the doctor who made it or by another doctor of our choice.

Having outlined the circumstances where a worker may have occasion to meet the doctor or nurse in the workplace I would like to deal briefly with an important issue, namely, the confidentiality of medical records in the occupational setting.

About 400 B.C. Hippocrates wrote the following oath which, to this day, is taken by physicians. ' . . . All that may come to my knowledge in the exercise of my profession or outside of my profession or in daily commerce with men, which ought not to be spread abroad, I will keep secret and will never reveal. If I keep this oath faithfully, may I enjoy my life and practise my art, respected by all men and in all times; but if I should swerve from it or violate it may the reverse be my lot.'

This oath relates to secrecy – the act of keeping to oneself what one has come to know. The patient's confidence that he or she may convey personal information to the doctor without fear that this will be

divulged to any one else is a fundamental *requirement*. Without this trust the physician's ability to assess the person and arrive at conclusions is severely compromised.

Are there instances where this requirement may not be assumed? There are some. In certain circumstances the doctor may be required to divulge secret information where it is required by law or when the right of society to be protected takes precedence over that of the individual patient.

A few examples are pertinent to illustrate the doctor's predicament. Consider the situation where a doctor examines another physician and finds that he or she has a mental illness which impairs his or her judgement. If the doctor concludes that this mental disorder could lead his patient to make serious errors in the treatment of patients, then he would be compelled to report this to the College of Physicians and Surgeons so they could investigate the situation and decide whether to allow the sick doctor to continue to practise. In this case, the right of the patients who could be injured takes precedence over the right of the sick doctor to secrecy. Think of the responsibility the examining doctor would have if he did not act.

Another common example is that of a doctor who examines a patient and notes increased eye pressure – tunnel vision. The doctor must report this to the department of transportation under the *Highway Traffic Act*. Once again, the rights of others are given precedence – but what consequences if there is failure to act!

Therefore, some sound advice. If you are planning to carry out a crime, don't discuss it with your doctor, or with your minister, for that matter. If asked, both must answer questions posed to them by the police regarding information you give them. Only a lawyer can refuse to provide such information.

Clearly, the situation is not as clean-cut as Hippocrates envisaged. This is especially true in the workplace. The fundamental principle remains – What is told to the doctor is between him and the patient unless the law overrules this or unless the patient *agrees* to let the doctor tell someone else. If the doctor is going to provide any information regarding the patient to a third party he should explain to the patient exactly what is going to be conveyed and, I believe, the patient should have the opportunity to read what will be sent and to approve it. Or if the information is to be conveyed verbally, the patient should have the option of being present at

the time that it is divulged and have the opportunity to intervene if information is being conveyed that the patient does not wish conveyed.

How might such a basic question arise? A common situation is in a fitness assessment. Here, the worker often consents to an examination as a condition of obtaining or retaining employment. The worker has a vested interest in being employed and in a job that suits him best – usually in that order of priority. The doctor wishes to ensure that the worker is fit for a job and will not pose a risk to himself or others. Unless the worker can talk to the doctor confidentially, trusting in the secrecy of what he says (unless it would harm others), it is in his best interest to tell nothing.

After protracted inquiry, in the instance of a pre-employment examination, Judge Horace Krever concluded that the doctor should decide two things as a result of his evaluation of an individual for a third party. He suggested that the doctor could conclude that the person was fit to work or not fit to work or fit to work with limitations. If the person is considered other than fit, the doctor should state why the person is not fit to work and ensure that the worker knows the reasons and understands why the doctor thinks there are limitations on his or her fitness. Before divulging any medical information about the patient to anyone, the physician should first ensure that the patient knows and understands what the other party is going to be told.

As you are aware, biological tests and medical examinations are required in the designated substances legislation under the *Occupational Health and Safety Act*. Chest x-rays are required for miners, and so on. These medical acts are a legislated condition of employment, and are in place primarily to protect workers from exposures that could damage their health. Such tests also give evidence of environmental conditions. Sometimes, even intensive environmental measurements, for instance in the case of lead, do not give adequate evidence of health risks. The medical acts are required because they give better evidence of environmental conditions.

The results of blood tests, under the designated substances legislation, are provided to worker, management and health and safety committees, clearly giving information about the worker to others even though no consent is obtained. Here is another example wherein the doctor obtains data, uses it to advise a worker, and has no option under the law but to provide the information to

a third party. The decision to allow this to happen is based on the need of the committee to have this information; it allows evaluation of work practices and protects the health of all workers in the plant. The public good supersedes the individual worker's right to privacy.

What if a doctor or nurse misuses the professional relationship with a worker by providing confidential information to a third party without the worker's consent?

The worker has protection from such acts of professional misconduct through the licensing and policing bodies of these two professions – the Ontario College of Physicians and Surgeons and the Ontario College of Nurses. Both have responsibility, delegated to them under the *Health Disciplines Act*, to investigate reported instances of professional misconduct of their members. If the doctor or nurse is found to have broken the confidentiality principle they can be fined, have their licence suspended for a period of time or the licence revoked by the regulation bodies, depending on the circumstances. If the worker is not satisfied with the professional colleges' evaluation, he or she may ask the Health Disciplines Board to intervene.

Am I suggesting you report physicians or nurses to their respective regulating bodies? Not necessarily. That decision is one which can only be made based on the circumstances which you or a fellow worker encounter. I have endeavoured to convey to you how the professional conduct of doctors and nurses is regulated to protect the public. Workers, like all patients, have a fundamental right to secrecy when it comes to what they tell a doctor or nurse. Medical and nursing professions stand behind this right and will take action against their members if it is not upheld. These professions cannot maintain their ethical principles unless you help them to become aware of instances where their members may not have lived up to the acceptable standards. Doctors and nurses are credible, trustworthy people. They need your help to ensure worker's confidence in them.

Another vital instance where confidentiality is often at issue is in the circumstance where a worker wants to know what substances, physical or chemical, he or she is working with. This is management's and government's responsibility. What adverse health effects may be expected is information the physician can discuss. Such information is often hard to understand, even for professionals. Workers must be able to trust these professionals to provide information, and they must also

rely on their own knowledge. You are as capable of understanding as the professionals, but through years of training they do have specific knowledge which can help you make fair assessments of such problems. It is no different from the previous situation where you must expect honest advice and interpretation of information in other health-related matters.

I appreciate this opportunity to address you on this topic which is important to your health, the health of your fellow workers and to your economic well-being. It is equally an important subject for the health professionals charged with the responsibility to promote and protect the health of all people in the workplace. Thank you.

Résumé

On traite de la responsabilité des docteurs et du personnel infirmier appelés à prendre des décisions pouvant affecter l'emploi et la possibilité d'emploi des travailleurs. On explore le conflit entre les droits du patient à la confidentialité et les besoins de renseignements d'une tierce partie concernant la santé du patient.

Fern K. Waterman, M.D., M.Sc.

Equilibrium of CO Between the Pregnant Mother and the Foetus

Summary

The uptake of carbon monoxide by women and its relevance with respect to the foetus is discussed. No threshold can be established below which there is a margin of safety for the foetus.

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The properties of carbon monoxide (CO) are well documented. Basically, it is a colourless, odourless, tasteless gas that is commonly formed during the incomplete combustion of carbonaceous material. It is sparingly soluble in water.[1] It exerts its toxic effects on mammals by competing with oxygen for haem compounds, particularly haemoglobin, to form carboxyhaemoglobin (COHb). The relative affinity of haemoglobin for CO as compared to that for oxygen in man is about 246 to one.[2] In having this greater affinity for the binding sites on the haemoglobin CO can thus seriously interfere with the oxygen carrying capacity of the blood. Furthermore, the carboxyhaemoglobin (COHb) interferes with the release of already bound oxygen at the tissue level, thus exacerbating an already anoxic situation.

Under changing CO exposures there is a complicated relationship between foetal and maternal COHb levels.[3] The foetus experiences a time lag of several hours in attaining equilibrium with the mother's COHb. CO is not very soluble in plasma,[1] and crosses the placenta by diffusion. Because of the maternal resistance afforded by the placental membranes *per se*,[4] as well as the poor solubility of CO, the half-time for the gas to diffuse across the placenta is about two hours. The time course of changes in foetal and maternal COHb with varying inspired CO concentrations is similar in humans[5,3] and sheep.[6] This was demonstrated by Longo and Hill,[6] using catheterized maternal-foetal vessels in pregnant sheep and their foetuses.

For obvious ethical and technical reasons, such studies on carbon monoxide exchange rates are impossible on human foetuses. However, there are well known relationships described[7,8] (Tables 1, 2 and 3) correlating the rate of changes in COHb levels to inspired CO concentrations, rate of activity and minute volume and endogenous production rate of CO for adults. Other authors[9,10,11,12] have documented the endogenous production of CO by the foetus. During a steady state the CO concentration in foetal blood should be slightly higher than that in maternal blood due to the effects of

both the endogenous CO production by the foetus and the diffusion of maternal CO across the placental membranes.[9] Furthermore, foetal haemoglobin has a greater affinity for CO than does maternal haemoglobin.[13]

Using this information, Hill, Hill and Associates[3] devised a mathematical model and calculated the theoretical relations of foetal/maternal COHb concentrations in human subjects. The resultant graphs resemble those derived from the data collected from the studies of pregnant ewes and their foetuses.[6]

Table 1

Expected COHb values under different conditions according to Peterson and Stewart (1975) at rest (alveolar ventilation: 6 l/min, diffusion capacity: 30 ml/min), light work (alveolar ventilation: 18 l/min, diffusion capacity: 40 ml/min) and heavy work (alveolar ventilation: 30 l/min, diffusion capacity: 60 ml/min). Height: 175 cm; weight: 75 kg (blood volume: approx 5.5 l), haemoglobin: 15 g/100 ml; barometric pressure: 750 mmHg; affinity constant for CO: 218; COHb.: 0.5%.[8]

duration	*200 ppm*			*100 ppm*			*75 ppm*			*50 ppm*		
	r	*lw*	*hw*	*r*	*lw*	*hw*	*r*	*lw*	*hw*	*r*	*lw*	*hw*
15 min	1.7	3.2	4.7	1.1	1.8	2.6	0.9	1.5	2.0	0.8	1.2	1.5
30 min	2.8	5.7	8.3	1.7	3.1	4.4	1.4	2.4	3.4	1.1	1.7	2.4
45 min	3.9	7.9	11.2	2.2	4.2	5.9	1.8	3.2	4.5	1.3	2.3	3.1
60 min	5.0	9.8	13.6	2.7	5.2	7.1	2.2	4.0	5.4	1.6	2.8	3.7
90 min	6.9	13.1	17.2	3.7	6.8	9.1	2.9	5.2	6.9	2.1	3.6	4.7
2 h	8.7	15.6	19.5	4.6	8.2	10.4	3.6	6.3	8.0	2.5	4.3	5.5
4 h	14.3	21.0	23.0	7.5	11.4	12.8	5.7	8.8	9.9	4.0	6.0	6.8
6 h	17.8	22.9	23.7	9.5	12.7	13.4	7.3	9.8	10.4	5.0	6.7	7.2
8 h	20.0	23.5	23.8	10.8	13.2	13.5	8.3	10.2	10.5	5.7	7.1	7.3
24 h	23.8	23.9	23.9	13.6	13.6	13.6	10.6	10.6	10.6	7.3	7.3	7.3

duration	*35 ppm*			*25 ppm*			*10 ppm*			*5 ppm*		
	r	*lw*	*hw*	*r*	*lw*	*hw*	*r*	*lw*	*hw*	*r*	*lw*	*hw*
15 min	0.7	0.9	1.2	0.6	0.8	1.0	0.5	0.6	0.7	0.5	0.5	0.5
30 min	0.9	1.4	1.8	0.8	1.1	1.4	0.6	0.7	0.8	0.5	0.6	0.6
45 min	1.1	1.7	2.3	0.9	1.3	1.7	0.6	0.8	0.9	0.6	0.6	0.6
60 min	1.3	2.1	2.7	1.0	1.6	2.0	0.7	0.9	1.0	0.6	0.6	0.7
90 min	1.6	2.6	3.4	1.3	2.0	2.5	0.8	1.0	1.2	0.6	0.7	0.7
2 h	1.9	3.1	3.9	1.5	2.3	2.9	0.8	1.1	1.3	0.6	0.7	0.7
4 h	2.9	4.3	4.9	2.2	3.1	3.5	1.1	1.4	1.5	0.7	0.8	0.8
6 h	3.6	4.8	5.1	2.7	3.5	3.7	1.3	1.5	1.6	0.8	0.8	0.8
8 h	4.1	5.1	5.2	3.1	3.7	3.8	1.4	1.6	1.6	0.9	0.9	0.8
24 h	5.3	5.3	5.3	3.9	3.9	3.9	1.6	1.6	1.6	1.0	1.0	1.0

r = rest
lw = light work
hw = heavy work

Table 2 Expected COHb values under different conditions according to Peterson and Stewart (1975) at light work (alveolar ventilation: 18 l/min; diffusion capacity: 40 ml/min) och COHb.: 5%. Height: 175 cm; weight: 75 kg (blood volume: approx 5.5 l): haemoglobin: 15 g/100 ml; barometric pressure: 750 mmHg; affinity constant for CO: 218.[8]

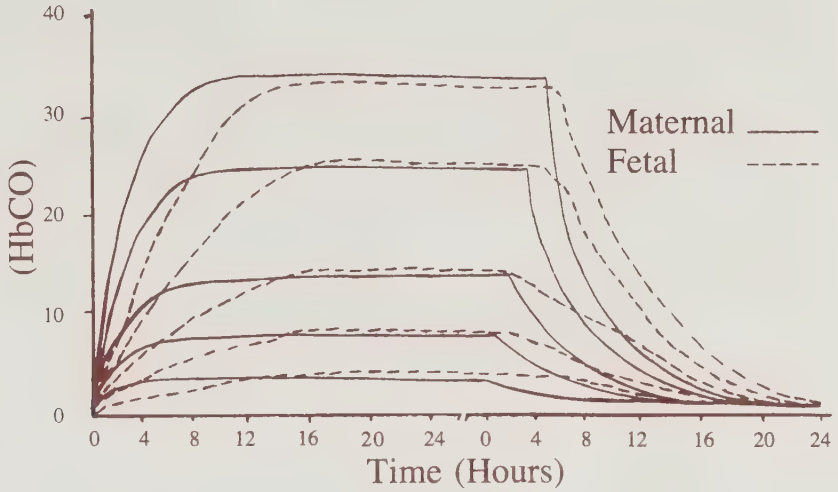
	200 ppm	*100 ppm*	*75 ppm*	*50 ppm*	*35 ppm*	*25 ppm*	*10 ppm*	*5 ppm*
15 min	7.3	5.9	5.6	5.2	5.0	4.9	4.7	4.6
30 min	9.3	6.7	6.1	5.4	5.1	4.8	4.4	4.3
45 min	11.1	7.5	6.6	5.6	5.1	4.7	4.1	4.0
60 min	12.7	8.1	7.0	5.8	5.1	4.6	3.9	3.7
90 min	15.3	9.2	7.7	6.1	5.1	4.5	3.5	3.2
2 h	17.4	10.1	8.2	6.3	5.2	4.4	3.2	2.8
4 h	21.6	12.2	9.6	6.9	5.2	4.1	2.4	1.8
6 h	23.1	13.0	10.2	7.1	5.3	4.0	2.0	1.3
8 h	23.6	13.4	10.4	7.2	5.3	3.9	1.8	1.1
24 h	23.9	13.6	10.6	7.3	5.3	3.9	1.6	0.9

Table 3 Expected COHb values under different conditions according to Peterson and Stewart (1975) at light work (alveolar ventilation: 18 l/min; diffusion capacity: 40 ml/min) for a small person (height: 165 cm; weight: 55 kg; blood volume: approx 4 l) with low haemoglobin value (11 g/100 ml). Barometric pressure: 750 mmHg; affinity constant for CO: 218; COHb.: 0.5%.[8]

	200 ppm	*100 ppm*	*75 ppm*	*50 ppm*	*35 ppm*	*25 ppm*	*10 ppm*	*5 ppm*
15 min	5.3	2.9	2.3	1.7	1.3	1.1	0.7	0.6
30 min	9.3	4.9	3.8	2.6	2.0	1.5	0.8	0.6
45 min	12.5	6.5	5.0	3.5	2.5	1.9	1.0	0.7
60 min	15.0	7.9	6.0	4.1	3.0	2.2	1.1	0.7
90 min	18.5	9.8	7.5	5.1	3.7	2.7	1.2	0.7
2 h	20.6	11.1	8.5	5.8	4.2	3.1	1.4	0.8
4 h	23.4	13.1	10.2	7.0	5.0	3.7	1.6	0.9
6 h	23.8	13.5	10.5	7.3	5.2	3.8	1.6	0.9
8 h	23.9	13.6	10.6	7.3	5.3	3.8	1.6	0.9
24 h	23.9	13.6	10.6	7.3	5.3	3.9	1.6	0.9

At all levels of CO exposure the maternal COHb concentration increases rapidly during the first four hours (see Figure 1). The changes in maternal COHb resemble a simple exponential process at this point, with a half-time of two to 2.5 hours. It then continues to increase more slowly over the next few hours, reaching a relatively constant level in seven to eight hours, and remaining so as exposure continues.

Figure 1



Maternal and foetal carboxyhaemoglobin concentrations during prolonged exposures to 30, 50, 100, 200 and 300 ppm inspired CO followed by a washout period when no CO is inspired. Note that foetal carboxyhaemoglobin concentrations lag behind mother's, but eventually reach higher values in most cases.[3]

The increase in *foetal* COHb levels lags behind maternal concentrations.[6] During the first hour of exposure foetal COHb concentration shows little change. During the following four to five hours it increases, but at a slower rate as compared with the rate of the earlier COHb rise of the mother. By 14 to 16 hours (24 hours for 300 ppm) foetal COHb equals maternal concentrations, after which it continues to increase slowly for 24 hours or more (i.e. it reaches the mother's level, but is still forming CO at its own endogenous rate as well). Only after 36 to 48 hours does it reach final steady-state COHb concentration at 15 per cent higher than the maternal concentrations. The time for foetal COHb concentration to reach half its final value at exposures of 100 ppm CO or less is about four hours.

After CO exposure ceases there is a washout phase, during which the foetal carbon monoxide elimination again lags behind that of maternal blood. This corroborates a study carried out in human mothers in their fifth or sixth month of pregnancy who were about to undergo legal abortion in Sweden, and who were exposed to varying levels of CO to raise maternal COHb concentrations to 15 per cent.[5] Blood COHb levels were then taken from both mother and foetus at the time of abortion, with each abortion performed at varying times – up to 40 hours after termination of the CO exposure.

During the washout phase the COHb concentration in the foetus is significantly greater than that in the mother. The time required to reach one-half of the final value here averages about two hours for the mother and seven hours for the foetus.

It has been reported that foetal COHb may be as great as 1.8 times as high as the maternal level,[14] a fact that could cause consternation regarding CO exposure in the workplace. The blood samples leading to this report, however, were taken from smoking mothers and their babies at the time of delivery.

The most common cause of increased CO inhalation and subsequent blood COHb concentrations from non-industrial indoor air is from tobacco use.

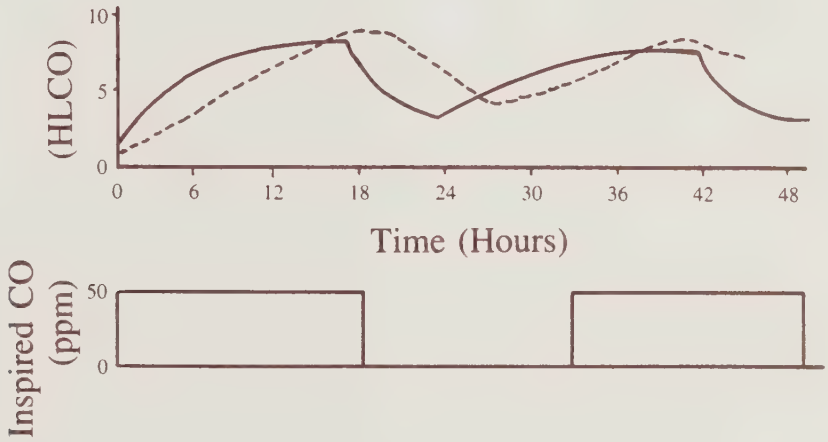
For pregnant mothers breathing relatively constant levels of CO, as in ambient air, the maternal COHb concentrations can range from 0.7 to 2.25 per cent in non-smokers[15,16,9,17] and up to 14 per cent in smokers.[18] Foetal COHb has been reported in the range of 0.44 to 2.02 per cent in non-smoking mothers[15,16,9,19] and up to 9.45 per cent in those delivered by smoking mothers.[14,30,21,9]

A non-smoking pregnant woman usually has a COHb level around 13 per cent higher than a non-pregnant woman, and under steady-state conditions the foetus has COHb concentration from 15 to 20 per cent above that of the mother.[9] This increase is a result of increased endogenous production of CO by the foetus itself.[10,12,22] Ratios of foetal COHb to maternal COHb have been reported ranging from 0.58 to 1.57[15,16,9,17] in steady-state conditions in non-smokers. Ratios from those in smoking mothers have been generally reported from 0.7 to 1.2[20,21,9] with a high of 1.8 reported by Cole and Hawkins, 1972.[14] This ratio is feasible under certain circumstances. However, samples taken from foetuses and their smoker mothers were not usually taken under steady-state conditions. They were taken at the time of delivery or as close to that time as possible (as were samples from non-smokers), with the last cigarette smoked by each mother at greatly varying times before delivery. The mother was therefore not exposed to a constant level of CO. The report 1.8:1 foetal COHb/ maternal COHb ratio is possible at the time of birth, depending on when the mother has smoked her last cigarette. This ratio is, very likely, not a reflection of what it had been throughout pregnancy as a steady state, but, rather, represents the foetal lag during the washout phase. Referring to Figure 1, if the mother's COHb had been within the smokers' range of five per cent, for

example, a level common amongst smokers, and if she had smoked her last cigarette $3^1/_2$ hours prior to delivery, her COHb level would then be half the value of her baby's. Although the neonate's COHb concentration would be double that of its mother, it would be on a downward trend at birth. If the infant remains unexposed to CO, there will be a steady decline in COHb concentrations until normal endogenous levels are attained.

Figure 2 shows results simulating COHb concentrations in a mother who smokes and in her foetus.[3] Although smoking patterns vary greatly among individuals, a 16-hour exposure to 50 ppm CO was chosen, followed by eight hours of breathing CO-free air, as a representative exposure. This might correspond to smoking about one and a half packs of cigarettes in a 16-hour period, followed by eight hours of sleep. According to the model, the foetal COHb concentration attains a peak somewhat higher and slightly later than the maternal peak. During the period of CO elimination, the foetal concentrations again remain higher than those of the mother, and do not have time to return to starting values.

Figure 2



Simulated carboxyhaemoglobin concentrations in a smoking mother and her foetus. Changes in $HbCO_m$ and $HbCO_f$ were calculated for a 16-hour exposure of 50 ppm inspired CO, which is about equivalent to smoking $1^1/_2$ packs of cigarettes per day, followed by an eight-hour sleep period.[3]

The COHb concentrations for a second period of exposure up to 50 ppm are also plotted. Again the peak foetal concentration is higher than the maternal, and occurs later.[3] The average COHb level over this period is six per cent in the foetus and 5.4 per cent in the mother. The

crossing of these curves suggests that one cannot estimate the foetal COHb on the basis of a maternal blood sample only, at least without knowledge of the exposure pattern.

Exposure of Pregnant Workers to Carbon Monoxide

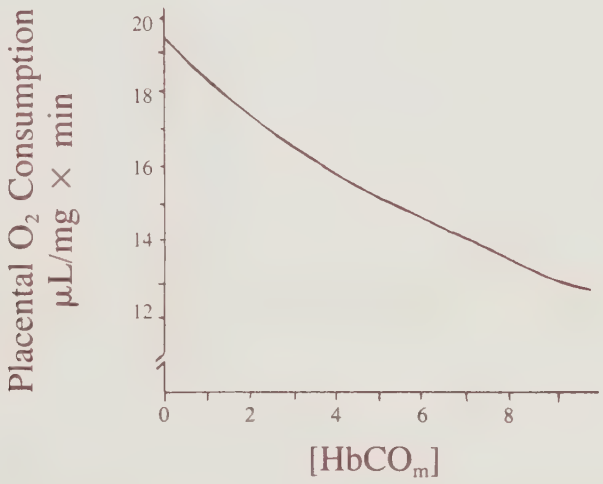
The Ministry of Labour has set a level of 35 ppm of CO as a time-weighted average exposure criterion, which is below the ACGIH TLV of 50 ppm,[1] to prevent possible cardiovascular and behavioural changes. As stated above, various investigators have documented and correlated exposure to CO, level of activity and blood COHb levels.[7,23,8] (Tables 1, 2 and 3), Exposure of pregnant workers to 35 ppm for eight hours while engaged in light work may give rise to COHb levels of 5.3 per cent. The blood COHb level would not rise beyond 5.3 per cent, even while engaged in heavy work for more than 24 hours. The time taken for foetal COHb to reach its final value exceeds eight hours, by which time the work shift would be over, and the foetal COHb of a non-smoking mother would therefore be under five per cent.

Smoking mothers may enter their occupational environment with a COHb concentration higher than 5.3 per cent. At this point the gas laws regarding equilibration prevail. If the worker's COHb is at a higher level than can be maintained by the ambient CO concentration and if she refrains from smoking during the shift, it will dissociate and begin to equilibrate with the surrounding air. The end result will be that the worker will expire CO. However, if smoking occurs during the shift the COHb level is usually maintained and is not affected by the lower concentration of CO in the workplace. Foetal COHb levels would therefore continue to be higher than those of the mother.

Problems may arise in that the foetal arterial blood PO_2 is normally only about one-third that of the mother's, (*20-30* mmHg, compared to the maternal 100 mmHg).[9] Because CO interferes with O_2 transport by displacing O_2 from haemoglobin in arterial blood, the blood oxygen transport capacity is compromised. To the pregnant woman, these effects on blood oxygenation may pose a special threat. Not only is her oxygen consumption increased 15 to 25 per cent during pregnancy,[19] but her blood oxygen capacity may be decreased 20-30 per cent or more because of a possible decreased haemoglobin due to anaemia. To date, it has been difficult to establish a threshold level of CO exposure to deal with this problem. There is some documentation available

from Tanaka.[24] It was reported in human placental studies[24] that oxygen uptake in tissue slices was impaired in smoking mothers. The oxygen consumption measured in placental slices from both smoking and non-smoking mothers varied inversely with the maternal COHb levels (Figure 3). For instance, among eight 'light' smokers (up to five cigarettes per day), placental oxygen consumption was reduced nine per cent, while it was decreased by 20 per cent in the placentas from nine 'heavy' smokers (more than five cigarettes per day). In four cases in which the COHb was more than seven per cent, mean placental oxygen consumption was 26 per cent less than normal values and 30 per cent less at eight per cent COHb in maternal blood. However, the blood levels of seven to eight per cent COHb in non-smokers should not be reached at an eight-hour or even 24-hour exposure to 35 ppm CO. Because of the time lag for foetal COHb to reach maternal levels, the pregnant worker will have left the workplace and will have had time to eliminate her CO accumulation. The half-time for elimination is about three to four hours when breathing air in a resting condition.[25,26,27]

Figure 3



Decrease in oxygen consumption by placental tissue as a function of maternal carboxyhaemoglobin concentration.[24]

The greater incidence of foetal death or gross foetal damage (mainly neurological), which has been reported, has occurred at CO exposures resulting in maternal COHb levels ranging from 20 percent to 95 per cent (i.e. from exposure to over 100 ppm CO for four hours to sudden acute exposure to several thousand ppm CO).[28,29,30,31] Similarly, the greater incidence of low birth-

weight babies[32,18,33] (200 gm or more below the average birth weight of 3,500 gm), spontaneous abortions, stillbirths and possible later learning problems in living children[34,35,36] associated with CO exposure occur in those children of mothers who often have COHb levels greater than those allowed by our current Ontario industrial criteria. These sequelae are associated with the COHb levels found in smoking mothers,[14,9,33] ranging from two per cent (under 10 cigarettes daily) to 14 per cent (over 30 cigarettes daily), with most studies reporting COHb blood concentrations of below six per cent. There are also the compounding effects of nicotine and other ingredients of cigarettes on placental circulation.[33] Thus the chronic 24-hour effect of smoking is different from that of intermittent occupational CO exposure in a healthy non-smoking mother.

Even in experimental studies conducted on animals exposed to CO,[37,38,9,6] teratogenic effects, stillbirths and small birth weights result from relatively high exposures (100 ppm to 1,500 ppm and higher).

In short, there seems to be no threshold that can be established below which there is a margin of safety for the foetus. Problems concerning maternal COHb levels and impaired oxygen consumption in humans seem to be at the chronic COHb levels associated with maternal smoking. A maternal blood level of six to seven per cent COHb could be attained at light work over six to eight hours (equivalent to walking a heavily travelled street [22] or heavy smoking in a non-ventilated room,[39] but should not be reached during a usual eight-hour 35 ppm TWA daily exposure in a non-smoker.

Résumé

On traite de l'absorption d'oxyde de carbone par les femmes et de son importance en ce qui concerne le foetus. On ne peut établir de seuil au-dessous duquel il existe une certaine marge de sécurité pour le foetus.

References

[1]American Conference of Governmental Industrial Hygienists, Inc. Documentation of the threshold limit values 1980; 4th. ed.

[2]Roughton FJW. The equilibrium of carbon monoxide with human haemoglobin in blood. Ann NY Acad Sci 1970; 174:177-188.

[3]Hill EP, Hill JR, Power GG, Longo LD. Carbon monoxide exchanges between the human foetus and mother: A mathematical model. Am J Physiol 1977; 232(3):H311-H323.

[4]Longo LD, Power GG, Forster RE. Placental diffusing capacity for carbon monoxide at varying partial pressures of oxygen. J Appl Physiol March 1969; 26(3):360-370.

[5]Friberg L, Nystrom A, Swanberg H. Transplacental diffusion of carbon monoxide in human subjects. Acta Physiol Scand 1959; 45:363-368.

[6]Longo LD, Hill EP. Carbon monoxide uptake and elimination in foetal and maternal sheep. Am J Physiol 1977; 232:H324-H330.

[7]Coburn RF, Forster RE, Kane PB. Considerations of the physiological variables that determine the blood carboxyhaemoglobin concentration in man. J Clin Invest 1965; 44:1899-1910.

[8]Peterson JE, Stewart RD. Predicting the carboxyhaemoglobin levels resulting from carbon monoxide exposures. J Appl Physiol 1975; 39(4): 633-638.

[9]Longo LD. Carbon monoxide in the pregnant mother and foetus and its exchange across the placenta. Ann NY Acad Sci 1970; 174:313-341.

[10]Maisels MJ, Pathak A, Nelson NM, Nathan DG, Smith CA. Endogenous production of carbon monoxide in normal and erythroblastic newborn infants. J Clin Invest; 50:1.

[11]Wranne L. Studies on erythrokinetics in infancy vii: Quantitative estimation of haemoglobin catabolism by carbon monoxide technique in young infants. Acta Paediatr Scand 1967; 56:381.

[12]Wranne L. Studies on erythrokinetics in infancy vi. A method for the quantitative estimation of pulmonary excretion of carbon monoxide in infancy. Acta Paediatr Scand 1967; 56:374.

[13]Longo LD. The biological effects of carbon monoxide on the pregnant woman, foetus and newborn infant. Am J Obstet Gynecol 1977; 129:69-103.

[14]Cole PV, Hawkins LH, Roberts D. Smoking during pregnancy and its effect on the foetus. Br J Obstet Gynaecol 1972; 79:782.

[15]Gemzell CA, Robbe H, Strom G. On the equilibration of carbon monoxide between human maternal and foetal circulation ''in vivo''. Scand J Clin Lab Invest 1958; 10:372-378.

[16]Linderholm H, Lundstrom P. Endogenous carbon monoxide production and blood loss at delivery. Acta Obstet Gynecol Scand 1969; 48:362-370.

[17]Young IM, Pugh GCE. The carbon monoxide content of foetal and maternal blood. Br J Obstet Gynaecol 1963; 70:681-684.

[18]MacMahon B, Alpert M, Salber EJ. Infant weight and parental smoking habits. Am J Epidemiol 1966; 82: 247-261.

[19]Pernoll ML, Metcalfe J, Schlenker TL, Welch JE, Matsumoto JA. Oxygen consumption at rest and during exercise in pregnancy. Respir Physiol 1975; 25:285-293.

[20]Haddon W, Nesbitt REL, Garcia R. Smoking and pregnancy: Carbon monoxide in blood during gestation and term. Obstet Gynecol 1961; 18:262-267.

[21]Heron, HG. The effects of smoking during pregnancy: A review with a preview. NZ Med J 1962; 61:545-548.

[22]Wright G, Jewczyk S. Onrot J. Tomlinson P, Shepard RJ. Carbon monoxide in the urban atmosphere: Hazards to the pedestrian and the street worker. Arch Environ Health 1975; 30:123-129.

[23]Peterson JE, Stewart RD. Absorption and elimination of carbon monoxide by inactive young men. Arch Environ Health 1970; 21:165-171.

[24]Tanaka M. Studies on the etiological mechanism of foetal development disorders caused by smoking habits during pregnancy. J Jpn Obstet Gynecol Soc 1965; 17: 1107-1114. English translation in J. Obstet Gynecol 1967; 14:45. (cited in Longo, 1977).

[25]Bartlett O. Pathophysiology of exposure to low concentrations of carbon monoxide. Arch Environ Health 1968; 16:719.

[26]Pace N, Strajman E, Walker EL. Acceleration of carbon monoxide elimination in man by high pressure oxygen. Science 1950; 111:652-654.

[27]Roughton FJW, Root WS. The fate of CO in the body during recovery from mild carbon monoxide poisoning in man. Am J Physiol 1945; 145:239.

[28]Breslau. Vier meitere beitrage zur gynakologie und geburstschulfe. Mschr Geburtsk Frauenkr 1859; 13: 435-456.

[29]Freund MB. Ein fall von absterben der frucht in ziebenten schwangerschafst monate in folge vier massiger. Intoxication der mutter durch kohlenoxydgas. Mschr Geburtsk Frauenkr 1859; 14:31-33.

[30]Goldstein DP. Carbon monoxide poisoning in pregnancy. Am J Obstet Gynecol 1965; 92:526-528.

[31]Tissier. Asphyxie par le gaz d'eclairage d'une femme au terme de sa grassusse. Obstetrique 1909;(2)14: 911-914.

[32]de Haas JH. Parental smoking: Its effect on foetus and child health. Eur J Obstet Gynecol Reprod Biol 1975; 5:283.

[33]Smoking and Health: A Report by the Surgeon General, 1979. U.S. Department of Health Education and Welfare, Public Health Service, Office of the Assistant Secretary for Health Office on Smoking and Health. DHEW Publication No. (PHS) 79-50066.

[34]Butler WR, Goldstein H. Smoking in pregnancy and subsequent child development. Br Med J 1973; 4:573.

[35]Dunn HG, McBurney AK, Ingram S, Hunter CM. Maternal cigarette smoking during pregnancy and the child's subsequent development. 11. Neurological and Intellectual Maturation to the Age of $6^1/_2$ years. Can J Public Health 1977; 68:43-50.

[36]Nichols PL. Minimal dysfunction: Association with perinatal complication. Paper presented at Society for Research in Child Development, New Orleans 1977.

[37]Astrup P, Olsen HM, Trolle D, Kjeldsen K. Effect of moderate carbon monoxide exposure on foetal development. Lancet 1972; 2:1220-1222.

[38]Baker FD, Tomasonis CF. Carbon monoxide and avian embryogenesis. Arch Environ Health 1972; 24:53-61.

[39]Russell MAH, Cole PV, Brown E. Absorption by non-smokers of carbon monoxide from room air polluted by tobacco smoke. Lancet 1973; 1:576-579.

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Frost Injuries of the Extremities*

Summary

Cold-injury to extremities as it relates to occupational exposure is discussed. Treatment and prevention of these injuries are considered.

Compared with other physical agents, such as heat, radiation and vibration, cold has received scant attention in occupational medicine. Major texts in the field either ignore it altogether[1] or give it very limited coverage.[2] Cold is an important occupational exposure and, as such, deserves more attention than it has received in the past. The purpose of this paper is to review freezing injuries of the extremities. Consideration will be given to who is occupationally exposed and the magnitude of the problem in Ontario. Mechanisms of injury, methods of treatment, and preventive strategies will also be discussed.

Occupational Exposure to Cold

The number of workers sufficiently exposed to cold to be at risk of developing injury is quite large. In the climate prevailing over much of Canada anyone working outdoors in winter might be considered at risk. This category would include letter carriers, trappers, forestry workers, fishermen, policemen and armed forces personnel. A smaller number of workers in cold indoor workplaces such as meat-packing plants and freezer plants are also at risk. Literature treating cold as an occupational exposure has come largely from the armed forces.[3,4,5]

Workers' compensation board figures may provide some estimate of the incidence of cold-induced injury. However, it must be realized that these figures are undoubtedly an underestimate of the number of cold-injuries. Only about 80 per cent of Canadian workers are covered by a compensation board[6] and the 20 per cent of the work force that is not covered is concentrated in occupations that put the workers at disproportionately higher risk of cold-injury.

Figures that are available indicate that more workers receive compensation for cold-related injury than for heat or sunstroke. The number of cold-injuries compensated in Ontario for selected years is given in Table 1.

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Table 1 **Cold Injuries Compensated in Ontario by Year** [7-10]

1976	112
1977	92
1978	61
1980	27

The temporal trend noticeable in the above Table may be due to differences in the severity of the winters rather than any improvements in preventive measures.

Acclimatization to Cold

Even allowing for under-reporting in compensation figures, cold-injury remains a fairly rare injury considering the number of workers at risk. There are probably several factors that help explain this. The injury usually is minor and those affected may recover fully without missing time from work. The cold-exposed person usually has advance warning of the exposure and dresses with the appropriate protective gear for reasons of injury-prevention and comfort. This is in marked contrast to many other hazards where the protective gear may be uncomfortable for the worker, leading to reduced compliance. In some cases it may also be possible to terminate exposure by leaving the cold area before injury ensues. A third way in which cold-injuries might be reduced is through acclimatization of those regularly exposed.

The subject of cold acclimatization is one on which there is a voluminous literature. Controversy in the field has revolved around such issues as the correct use of the terms adaptation, acclimatization and acclimation, and, regardless of the term used to describe it, whether there is any response similar to that seen in repeated exposure to heat. Goldsmith's[11] definition of acclimatization: 'Those changes in the responses of an organism produced by continuous alterations in the environment' will be used for the purposes of the following discussion. There are two main types of acclimatization to cold discussed in the literature. The first is that of a general metabolic change in how the body responds to cold. This would require some means of increasing metabolic heat production in addition to the shivering that is found in non-acclimatized subjects. Evidence for the existence of such a phenomenon

has been found in rats, but evidence in humans is much weaker. Studies have failed to demonstrate cold acclimatization in Inuit, but possible acclimatization has been found in Kalahari bushmen and Korean skin divers. However, even in these cases metabolic rate is not increased and thus nothing comparable to the non-shivering thermogenesis of rats is found.

The other possible acclimatization mechanism revolves around keeping heat in. Humans exposed to polar conditions may insulate themselves with a thicker layer of subcutaneous fat. Another method of conserving heat is by a vascular response to cold in the extremities. The usual response to plunging a hand into cold (4°C) water is an increase in blood pressure, increased heart rate and vasoconstriction; all of which are mediated by increased sympathetic activity. The increased blood pressure and heart rate does impose a stress on the body. LeBlanc[12] has shown that Inuit respond with less sympathetic activity than non-acclimatized Caucasians to this cold challenge.

The shunting of blood from the extremities and skin to conserve heat makes good sense to the homeotherm, and it is not difficult to understand why this has become built in as a reflex. For purposes of heat conservation, homeotherms may be thought of as comprising a core and a periphery. It is really the core temperature that is maintained and defended against changes that may be caused by the environment. The skin and, to a lesser extent, the extremities are poikilothermic structures as their temperature is not maintained nearly as rigorously as that of the core. For example, the skin of the foot may drop 7°C in response to an environmental temperature change from 30°C to 20°C, while the skin of the forehead drops only 1°C.[13] Furthermore, the steady-state difference between foot and forehead skin temperature increases with decreasing ambient temperature.

At some point the benefits of heat conservation by allowing extremity temperature to drop will be outweighed by the local tissue damage due to cold in the extremity. A defence against this has been built into the body in the form of a local cold-induced or 'paradoxical' vasodilation. This occurs when skin temperature falls below 15°C and provides some protection from frostbite. If the cold-exposure persists, the body attempts to maintain a balance between central and peripheral needs with alternating periods of vasoconstriction and vasodilation. Obviously, this defence mechanism can only protect the body up to a certain point, beyond which deleterious effects will ensue. Whether or not tissue

damage will occur depends on a number of individual and environmental factors. On reviewing studies of finger freezing, Wilson[14] concluded that there was no clear value of the wind-chill index above which frostbite could be predicted with certainty, and that frostbite in acute exposures seldom occurs at air temperatures above −5°C, even with a strong wind.

Types of Cold Injury

Cold-injuries may be classified in several different ways and with varying detail. Basically, they are of two types: freezing and non-freezing. The former includes frostnip and frostbite of various degrees of severity; the latter, trench and immersion foot. Trench foot and immersion foot are not seen regularly in civilian practice and will not be discussed further. Frostnip is the most minor of the freezing injuries. It often involves the ear lobes or the tip of the nose and is of so little significance that it only requires exposure to warm air or warmth from some other part of the body to be fully reversible. Frostbite severity is indicated by degrees 1 through 4. According to Killian[15] frostbite passes through four phases: the actual cooling phase, the rewarming phase, the phase of reactive hyperemia and the reparative phase. The severity of a frostbite injury may not be obvious initially and may be classified as outlined in Table 2.

Table 2 **Severity of Frostbite**

Degree	*Damage*
1	Oedema and redness without necrosis
2	As above, but with blisters
3	Skin necrosis
4	Gangrenous; some amputation

Pathophysiology of Frostbite

The tissue damage that occurs in frostbite may be attributable to two major mechanisms.[16] The first is ice formation in the tissue with its related osmotic effects. Ice crystals may form in the interstitial or intracellular fluid and directly damage cellular structures or damage may be caused indirectly by osmotic effects. In slow

freezing, ice crystals will form first in the interstitial fluid; the osmotic pressure of the extracellular fluid will increase, withdrawing water from inside cells. The intracellular milieu becomes hyperosmolar and cell death may ensue.

Vascular stasis is the second major mechanism by which frostbite may cause tissue damage. With cooling, blood circulation slows and eventually stops. Without the arrival of oxygen and nutrients and the removal of wastes cellular activity grinds to a halt. Circulation may be further compromised by capillary damage.

Treatment

In the past, controversy has centred on whether frostbitten parts should be rewarmed rapidly or slowly[17] as well as the means that should be used to effect the warming. A consensus seems to have emerged in favour of rapid rewarming.[18-22] The best method is immersion of the affected part in warm water that is maintained at 38°C to 42°C. Water hotter than this is likely to be harmful and may add a heat injury to the one already existing from cold-exposure. Thawing should not be attempted in the field if there is any chance the tissue will be allowed to refreeze before proper treatment facilities are reached. The freeze, thaw, refreeze sequence seems to be more harmful to tissue than extending the time of the initial freeze. Frozen extremities should not be massaged or have snow rubbed on them as this is also likely to add further injury to traumatized tissue. Complete thawing of the extremity usually takes less than 30 minutes with immersion in water, whereas thawing by other means may require three to four times as long. After thawing, the affected area will become painful, hot, red and oedematous. This is probably due to capillary damage with attendant leakage of fluid into the interstitial space.

The damaged area should be examined and cleaned. If tetanus status is in doubt, a booster should be given. If the area cannot be cleaned properly or if there are other reasons for believing that infection may develop, broad-spectrum antibiotics may be used. Analgesics sufficient to control pain should be given and ASA-containing compounds may be especially beneficial through their inhibition of platelet stickiness. Unbroken blisters should be left intact; loose, dry, sterile dressings should be applied and arrangements made for suitable follow-up.

Severe cases may require hospitalization. Early operative intervention is to be discouraged as, often,

injuries will prove to be far less extensive than originally thought. Gangrene may be confined to superficial tissue and, with the passage of time, fall away revealing viable tissue underneath.

Sequelae to frostbite may include increased future susceptibility to cold injury, recurrent pain, paresthesias and hyperhidrosis. These may be due to permanent local changes or to an increase in vasomotor tone due to the cold-exposure.

Prevention

While effective treatment of cold-injury can be given in most cases, the emphasis should be on prevention. Measures for preventing cold-injuries are similar to those used for preventing other occupational illnesses and injuries, although the relative emphasis placed on various measures may vary.

The armed forces have paid much attention to the effects of cold and have developed strategies for minimizing its effects. The impetus for this has stemmed from the fact that military commanders must maintain their men at peak efficiency under prolonged adverse conditions in times of war. However, even under peacetime conditions, military physicians may receive requests for advice regarding allowable cold-exposure for personnel. Although no cold weather exposure limits have been regulated, those in Table 3 have been recommended in the Canadian forces.

Table 3 **Suggested Cold-Exposure Limits**

Temperature Range	*Maximum Daily Exposure*
30°F – 0°F (−1°C to −18°C)	No limit to exposure time if the person is properly clothed
0°F – −30°F (−18°C to −34°C)	Total cold work time: four hours. Alternate one hour in and one hour out of environment
−30°F to −70°F (−34°C to −57°C)	Two periods of 30 minutes each at least four hours apart. Total cold environment work time allowed: one hour
−70°F to −100°F (−57°C to −73°C)	Maximum permissible cold-environment work time: five minutes over an eight-hour working day

In its attempt to understand cold-injuries, the military has employed the host, agent, environment triad from classical epidemiology. The agent, cold, while clearly of etiologic significance, is subject to several modifying factors, including humidity and wind velocity. The distinction between environment and agent factors is somewhat blurred in the case of cold, but takes into account the circumstances under which the exposure takes place as this may also play a part in the ultimate severity of any injury. In military terms a distinction may be made between troops engaged in offensive and defensive manoeuvres. In civilian settings distinctions might be drawn between intermittent exposures with frequent opportunity to rewarm versus the unremitting exposure that might be experienced by a fisherman or a trapper.

There are, undoubtedly, some individuals at higher risk of developing cold-induced injury than others. A list of medical conditions that pre-dispose to the development of peripheral cold-injuries is given by Killian.[15] These are presented in a modified form in Table 4.

Table 4 **Conditions Which Pre-Dispose to Peripheral Cold-Injury**

Buerger's disease (thrombo-arteritis obliterans)
Arteriosclerosis
Previous cold-injury
Giant cell arteritis
Traumatic vascular damage
Raynaud's disease

Given the nature of the hazard, it may not be advisable to rigidly eliminate anyone with one of the above conditions from cold-exposure. But pointing out to people, on an individual basis, that they are at higher risk of injury may raise their awareness of the possibility of cold-injury and increase the likelihood that they will take appropriate preventive measures.

Other host variables have also been considered as potentially important in the development of cold-injuries. Age, within the range found in the working population, is not thought to be important, although the very young and the elderly may be more susceptible. Previous cold-injury or concomitant traumatic injury may place people at higher risk of injury during cold-exposure. Use of nicotine or alcohol by persons exposed to cold is

not recommended. Nicotine, due to its vasoconstricting action, may result in more severe injury than would have otherwise occurred. Alcohol, while potentially increasing blood flow to the skin or extremities, can cause increased heat loss from the body and thus increase the risk of hypothermia. Similar effects may be produced by other drugs, and the potential for a harmful side-effect under cold conditions should be borne in mind by physicians.

Nutritional status of cold-exposed people is probably not of importance unless the person is in a starved or semi-starved state.

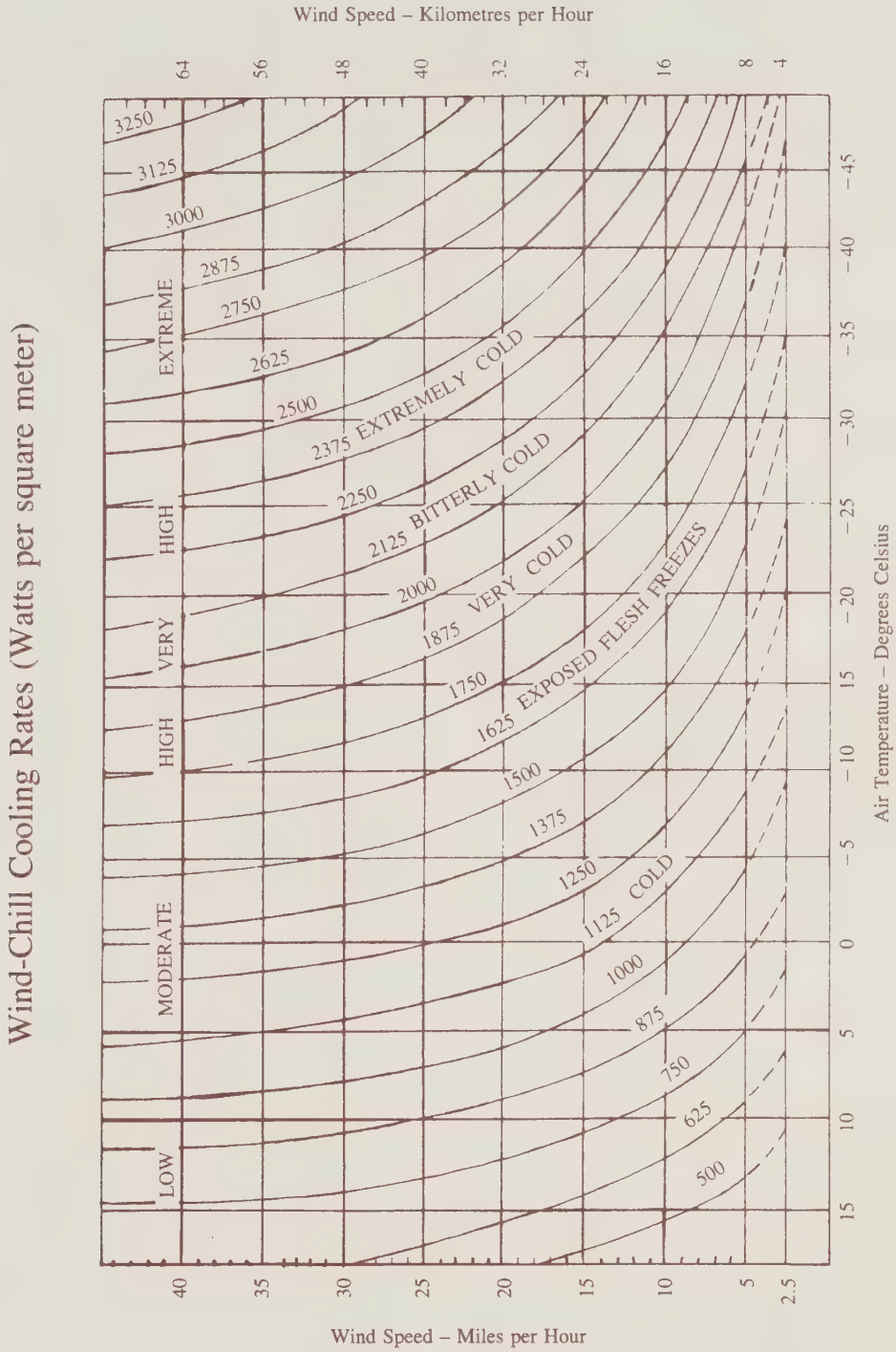
Racial differences in susceptibility to cold-injury have been postulated, based largely on the high frostbite-injury rate observed in black troops in the U.S. Armed Forces in World War II and Korea. Whether this represents a *bona fide* racial difference or whether it is due to differences in education and past familiarity with cold has not been resolved. In at least one instance, workers for projects in the far North have been selected on the basis of their ethnic background and the location where they were raised.[2] Firm data on whether this was of benefit are lacking.

Several measures have been suggested by armed forces' authors for preventing cold-injuries; many of these are applicable to civilian workers as well. An important step in preventing cold-injuries is to reinforce in everyone's mind the fact that cold-injuries are preventable if proper precautions are taken.

Awareness of weather conditions is a must; managers and employees should realize that, under certain conditions, outside work may have to be curtailed. The armed forces' guidelines in Table 3 do not take into account the effect of the wind-chill factor. Wind-chill cooling rates are provided in Figure 1. Important points on the wind-chill continuum are: 1,600, where freezing of exposed skin begins (this is affected to some extent by activity level and the presence or absence of sunshine); 2,300, conditions for outdoor travel become dangerous, exposed flesh may freeze in less than one minute; 2,700, exposed flesh freezes in 30 seconds.

Large groups of cold-exposed workers (15 or more) should have someone designated to look after preventive measures and monitor compliance with proper practices by others. Individuals should be encouraged to observe co-workers for early signs of frostbite (e.g. blanching). In the earliest stages, touching (not rubbing) a warmer part of the body to the just-frozen area may be sufficient

Figure 1



to reverse cold-induced changes. Afterwards the part must, obviously, be protected from further serious exposure to cold. Where possible, rewarming facilities should be provided for workers. Activities under cold conditions must be paced to minimize the chance of cold-injury. The activity level should be sufficient to maintain good circulation in all extremities, but not so great as to cause sweating, which may lead to increased

heat loss with moisture trapped in various layers of clothing. Where possible, activities should be carried out in areas protected from the wind. In some jobs it may be possible to provide workers with a shelter or a windbreak.

Proper clothing is essential to preventing cold-injury. In general, several layers of light garments are far more effective than one heavy layer. This is due to the insulating effect of the air trapped between the layers. Obviously, if the wearer perspires, and the clothes are no longer dry, the insulating effect is lost. For the same reason care must also be taken to keep the clothing free from external sources of moisture. Clothing should be loose enough that circulation is unimpaired. It is hazardous to force extra insulating material into an already tight article of clothing if this compromises blood-flow. These principles have been understood by aboriginal peoples inhabiting far northern regions, and some feel that modern cold-weather garments, including those used by the armed forces, may not be as effective as those used by aboriginals.[23]

As the face, hands and feet are at the highest risk of developing frost injuries, extra care should be taken in protecting these parts of the body. A lined hood provides good protection for the face and ears. Some trade-off may have to be made between the protection afforded by a hood and its reduction of the field of vision.

Protection of the feet is of great importance. Boots should be warm and waterproof; multiple sock layers may be used. Sweating from the feet can pose some problems. For this reason, if prolonged cold-exposure is anticipated, extra socks should be carried so that moisture-soaked ones can be changed when the need and opportunity arises. Inactivity and maintenance of the foot in a dependent position may also predispose to injury. Some armed-forces authorities recommend vigourous hourly activity sessions for sedentary troops at temperatures lower than $-20°F$ ($-29°C$). This activity should not, however, be carried to the point of perspiration.

Hands are also susceptible to cold-injury. Again, the general principles of multiple layers of clothing and avoidance of constricting articles apply. Mitts offer more protection than gloves. If mitts do not allow enough dexterity for certain tasks, a glove can be worn underneath. The mitt can be removed for tasks that require a gloved hand and replaced when the task is

completed. If machinery must be handled bare-handed for brief periods at low temperatures, care should be taken that metal surfaces are not touched. It is not only the absolute temperature of an object that is important, but its thermal conductivity. The sensation of object-temperature is related to this latter property because materials with a high thermal conductivity are much better at removing heat from the skin. The conductivity of steel is about four times that of rubber and over 80 times that of cork.[13]

Conclusion

Occupational exposure to cold can result in injury to extremities, usually minor in nature, but with some potentially serious results. As with other occupational exposures, the key lies in preventing the injury. Heightened awareness of the problem may contribute to its solution.

Résumé

On traite des blessures aux extrémités dues au froid se rapportant à l'exposition au froid pendant le travail. On examine le traitement et la prévention de ces blessures.

References

[1]Hunter D. The Diseases of Occupations. 6th ed. London: Hodder and Stoughton, 1978.

[2]Butan RC. Work under low temperatures and reactions to cold stress. In: Zenz C. Occupational Medicine: Principles and Practical Applications. Chicago: Year Book, 1975: 493.

[3]Varma RN, Boparai MS. Cold Injury as an Occupational Hazard in the Armed Forces. Indian J. Med. Reg. 1974; 62: 1478-1485.

[4]Dembert ML, Dean LM, Noddin EM. Cold Weather Morbidity among United States Navy and Marine Corps Personnel. Milit Med 1981; 146: 771-775.

[5]Vaughn PB. Local cold injury – menace to military operations: a review. Milit Med 1980; 145(5): 305-11.

[6]Labour Canada. Canadian Employment Injuries and Occupational Illness, 1979 ed.; Ottawa: Labour Canada.

[7]Ontario Ministry of Labour. Advisory Council on Occupational Health and Occupational Safety, 1st Ann. Rep., April 1, 1978 to March 31, 1979: 111.

[8]Ontario Ministry of Labour. Advisory Council on Occupational Health and Occupational Safety, 2nd Ann Rep, April 1, 1979 to March 31, 1980: 384.

[9]Ontario Ministry of Labour. Advisory Council on Occupational Health and Occupational Safety, 3rd Ann Rep, April 1, 1980 to March 31, 1981: 284.

[10]Ontario Ministry of Labour. Advisory Council on Occupational Health and Occupational Safety, 4th Ann Rep, April 1, 1980 to March 31, 1981: 224.

[11]Goldsmith R. Acclimatisation to heat and cold in man. In: Bacon DN, Compston N, Dawson AM, eds. Recent advances in medicine, Vol. 17. Edinburgh: Churchill Livingstone, 1977: 299-321.

[12]LeBlanc J. Evaluation of adaptation to polar environment by autonomic nervous system responses. In: Edholm OG, Gunderson GKE, eds. Polar Human Biology. Chichester: Heinemann, 1973: 256-264.

[13]Houdas Y, Ring EFJ. Human Body Temperature, Its Measurement and Regulation. New York: Plenum, 1982.

[14]Wilson, O. Experimental Freezing of the Finger: A review of studies. In: Edholm OG, Gunderson EKE, eds. Polar Human Biology. Chichester: Heinemann, 1973: 246-255.

[15]Killian H. Cold and Frost Injuries. Berlin: Springer-Verlag, 1981.

[16]Gage AM, Gage AA. Frostbite. Comp Ther 1981; 7(9): 25-30.

[17]Petersdorf RG. Disturbances of Heat Regulation. In: Thorn GW, Adams RD, Braunwald E, Isselbacher KJ, Petersdorf RG. Harrison's Principles of Internal Medicine. 8th ed. New York: McGraw-Hill 1977: 53-59.

[18]Berkow R, ed. Cold Injury. In: The Merck Manual of Diagnosis and Therapy. Rahuay, N.J.: Merck Sharp Dohme, 1977: 1727-1729.

[19]Cain HD, ed. Temperature variation emergencies. In: Flint's Emergency Treatment and Management. 6th ed. Philadelphia: Saunders, 1980: 667-675.

[20]Anon. Treatment of Frostbite. Med Lett Drugs Ther 1980; 22(26): 112-114.

[21]Miller BJ, Chasmor LR. Frostbite in Saskatoon: A review of ten winters. Can J Surg 1980; 25: 423-426.

[22]U.S. Navy. Flight Surgeon's Manual. 2nd ed. Washington: U.S. Government Printing Office, 1978.

[23]Tepper ML. Canadian Forces Base, Edmonton: unpublished memorandum.

[24]Rode A. Cold: Special Aspects in the Canadian Environment. Ottawa: National Research Council, 1983.

Peter J. Howe and Robert Kusiak

County Level Standardized Mortality Ratios in Ontario, 1954-1978

Summary

Standardized mortality ratios are presented for the county/district/regional municipality level in Ontario in five-year calendar periods for the years 1954-78 for a number of groups of causes of death.

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Standardized mortality ratios in various geographically defined populations in Ontario have been calculated in the Special Studies and Services Branch of the Ontario Ministry of Labour for a number of years. These ratios have been helpful in determining whether mortality rates in a specific group of people vary significantly from the average mortality rates in the province.

This paper attempts to present a more comprehensive historical review of various causes of mortality in the province than is available from other sources.

Standardized mortality ratios are used because they are easily calculated from the data available and they produce an estimate of the average relative difference between the mortality rates in the municipality and the province as a whole.

Data

Mortality data for all counties and selected cities, towns, villages and townships are published annually by the Ontario Registrar-General.[1] These reports give the number of deaths classified by place of residence, sex and List A, the Intermediate List of 150 Causes for Tabulation of Morbidity and Mortality of the International Classification of Diseases (ICD). The publications also contain Tables of all Ontario residents' deaths classified by age, sex and List A of the International Classification of Diseases.

Three versions of the ICD codes have been used between 1954 and 1978. In order to make the cause-specific ratios consistent between time periods, some adjustments were made to the groupings of the causes.

Statistics Canada publishes the population counts made in each census.[2-6] These are classified by sex, age group and geographic place of residence. Census data are collected every five years.

Analysis of Data

The Standardized Mortality Ratio (SMR) is a summary statistic that is used to compare average mortality rates in the county with the average mortality rates in the province, and removes the confounding effect of differences between the age distributions of the province and the county by adjusting to the age distribution of the county. SMRs for a particular calendar period are calculated as follows:

$$SMR = 100(O/E)$$

where $E = \Sigma j\ (p_j d_j)$

- O = Observed number of deaths in the county during the calendar period
- E = Expected number of deaths in the county during the calendar period
- p_j = Census population count of the jth age group in the county at the mid-point of the calendar period
- d_j = provincial death rate in the jth age group during the calendar period

The expected number of deaths gives the average number of deaths that would occur in the county if the age-specific rates in the province and the county were equal. The SMR is then used to compare the observed mortality with the mortality that would have occurred if age-specific rates in the county and the province were the same.

Large SMRs tend to indicate that the county rates are higher than provincial rates, while small SMRs tend to indicate the opposite. The probability that the difference between observed and expected values is due solely to chance variation can be calculated. For small numbers of observed and expected deaths the Poisson distribution can be used, while for larger numbers the approximation given by the normal distribution can be used. When the observed and expected numbers are larger than 20, the test statistic is given by $(O\text{-}E)^2/E$. If the assumption of no difference between observed and expected is true, the test statistic has a chi-square distribution with one degree of freedom. If there is no difference between observed and expected numbers, the test statistic will be larger than 3.84 for up to five per cent of the time, larger than 6.635 for one per cent of the time and larger than 10.828 for 0.1 per cent of the time. The statistical logic says that when the test

statistic is larger than 3.84, the probability that the difference is due to chance alone is small, and one concludes that there is a true difference between the county and provincial rates, and the chance that this conclusion is false is five per cent.

This logic is entirely appropriate when one proceeds on the basis of some information that does not relate to mortality in the given county. The logic breaks down, however, if one were to scan through the attached Table and select high or low SMRs and then proceed to calculate a probability level in order to see if the difference between a county with a high or low SMR and the province as a whole is 'significant'. In this case, the resulting value does not indicate the probability that the difference between observed and expected is due to chance alone. The logic is faulty because the calculation of the 'probability' was suggested by the size of the SMR.

There is one well-known technique that can be used in order to produce a valid probability statement about the SMR for a given county, and the technique depends upon the person viewing the results of the survey. The technique is to adjust the probability level that we consider small by dividing the usual five per cent level by the number of SMRs that were being scanned. For example, if one were interested only in, say, male lung cancer rates averaged over the years 1954 to 1978, then the number of SMRs being scanned is the number of counties, 47, and the usual significance level should be adjusted to $0.05/47 = 0.001$. Only those SMRs for which the test statistic exceeds 10.828 should be considered as significant, and the risk of being wrong in making that conclusion would still be about five per cent or less.

Presenting the data in this way invites the reader to compare SMRs in two different counties.

A word of caution is necessary. It is well-known that the value of an SMR may depend upon the age distribution of the county population.[7,8] The difficulty arises when the increased risk of mortality varies with age and the two counties have different age distributions. Clearly, then, the SMRs of the two counties will differ, although the same risk is present in both counties. The following example illustrates this effect. Suppose that there are two counties, each with 1,000 persons. Suppose also that there are two age groups, A and B, and the death rate is such that 10 per cent of Group A and Group B are expected to die. Assume that in each

county, the mortality rates are elevated above the provincial rate by 10 per cent in Group A and 20 per cent in Group B. The following calculation shows that the SMR in County 1 will be 116, while in County 2 the SMR will be 114. The difference in the values of the SMRs is due to the difference in the age structures of each population.

Effect of Age Distribution on Value of SMR

	County 1		*County 2*	
Age Group	A	B	A	B
Population	400	600	600	400
Expected Deaths	40	60	60	40
Observed Deaths	44	72	66	48
SMR	$100\frac{(44+72)}{40+60}$		$100\frac{(66+48)}{40+60}$	
	$= 116$		$= 114$	

Results

The following Table contains the observed and expected number of deaths from, and SMRs for, all malignant neoplasms, diseases of the circulatory system, diseases of the respiratory system, and violence.

There are 376 SMRs presented in the Table and, in order to achieve an overall significance level of five per cent in a two-sided test, each SMR was tested at the 0.01 per cent level. Significant SMRs are shown with an asterisk.

For mortality due to malignant neoplasms, the SMRs are significantly elevated in males and females in Metropolitan Toronto, including York Region, and in males in Hamilton-Wentworth and Niagara. SMRs are significantly low in males in Bruce, Durham, Grey, Huron, Muskoka, Northumberland, Oxford, Parry Sound, Perth, Prescott and Russell, Prince Edward, Renfrew, Simcoe, Waterloo and Wellington. None of the SMRs for females are significantly low.

SMRs for diseases of the circulatory system are significantly high in males and females in Algoma, Essex, Nipissing, Simcoe, Sudbury, Thunder Bay and Temiskaming. They are significantly low in males and females in Peel, and York and Metropolitan Toronto. They are significantly low in males in Halton, Kenora, and

Lennox and Addington, and in females in Middlesex. They are significantly high in males in Ottawa-Carleton, and in females in Cochrane, Haldimand-Norfolk, Lambton, and Prescott and Russell counties.

SMRs for diseases of the respiratory system are high in males and females in Cochrane, Frontenac, Hastings and Kenora. They are significantly high in males in Middlesex, Thunder Bay and Temiskaming, and in females in Lennox and Addington, Parry Sound, Rainy River, Renfrew, and Stormont, Dundas and Glengarry. They are significantly low in both sexes in Peel, Waterloo and Wellington. They are significantly low in males in Bruce, Durham, Grey and Halton, and in females in Elgin and Metropolitan Toronto, including York Region.

SMRs for violent causes of death in males and females are low in Hamilton-Wentworth, Waterloo, and York and Metropolitan Toronto, and are high for males and females in Cochrane, Kenora, Manitoulin, Nipissing, Parry Sound, Sudbury, Thunder Bay and Temiskaming. They are significantly high in males in Algoma, Bruce, Dufferin, Grey, Haldimand-Norfolk, Haliburton, Hastings, Huron, Lambton, Muskoka, Northumberland, Oxford, Prescott and Russell, Rainy River, Renfrew, Simcoe, and Stormont, Dundas and Glengarry, and significantly low in males in Halton, Niagara, Ottawa-Carleton and Peel.

Additional Tables showing other causes of death are available from the authors.

Mortality Data by County 1954-1978

County	Sex	Malignancies			Diseases of Circulatory System			Diseases of Respiratory System			Accidents, Poisonings and Violence		
		Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR
Algoma District	M	1897	1764.7	107	5592	5049.6	111*	753	662.9	114	1827	1201.3	152*
	F	1220	1261.5	97	3486	2991.4	117*	358	340.6	105	521	450.4	116
Brant County	M	1906	1930.4	99	6093	5869.3	104	830	740.2	112	953	1000.7	95
	F	1664	1689.7	98	5257	5067.1	104	558	494.3	113	478	477.8	100
Bruce County	M	1025	1351.4	76*	4284	4318.8	99	448	544.7	82*	718	566.9	127*
	F	949	984.6	96	3390	3165.4	107	301	300.1	100	291	263.3	111
Cochrane District	M	1807	1665.3	109	4937	4710.3	105	967	614.9	157*	1752	1040.5	168*
	F	1075	1010.0	106	2534	2159.6	117*	357	256.2	139*	486	363.6	134*
Dufferin County	M	400	483.1	83	1469	1506.7	97	161	189.3	85	291	227.3	128*
	F	401	371.5	108	1230	1176.4	105	106	113.1	94	125	104.9	119
Durham Region	M	3357	3734.0	90*	10739	11125.3	97	1254	1434.2	87*	2038	2227.6	91
	F	2853	3044.1	94	8488	8540.8	99	816	876.0	93	990	969.8	102
Elgin County	M	1438	1581.1	91	4894	4964.1	99	567	617.6	92	838	740.9	113
	F	1233	1348.0	91	4161	4318.4	96	314	411.3	76*	406	367.1	111
Essex County	M	6028	5858.1	103	18124	17314.4	105*	2283	2215.6	103	2902	3100.1	94
	F	4539	4675.8	97	13106	12505.7	105*	1277	1254.8	102	1361	1351.8	101
Frontenac County	M	1630	1755.5	93	5136	5281.5	97	907	678.4	134*	993	1062.2	93
	F	1480	1537.3	96	4421	4567.9	97	675	459.8	147*	434	470.9	92
Grey County	M	1475	1822.8	81*	5571	5787.3	96	588	723.1	81*	929	776.1	120*
	F	1343	1404.6	96	4482	4453.5	101	374	421.9	89	370	371.9	99
Haldimand-Norfolk Region	M	1803	1932.5	93	6001	5873.4	102	667	742.9	90	1262	936.4	135*
	F	1434	1462.1	98	4501	4193.3	107*	434	412.1	105	442	413.1	107
Haliburton Provisional County	M	234	266.0	88	722	784.7	92	86	98.0	88	156	106.7	146*
	F	140	174.2	80	444	445.2	100	40	43.3	92	56	43.8	128
Halton Region	M	2133	2136.9	100	5534	5879.5	94*	630	785.2	80*	1272	1513.3	84*
	F	1917	1811.6	106	4393	4443.4	99	416	488.7	85	571	633.8	90

County	Sex	Malignancies			Diseases of Circulatory System			Diseases of Respiratory System			Accidents, Poisonings and Violence		
		Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR
Hamilton-Wentworth Region	M	7889	7326.7	108*	21287	21555.2	99	2852	2727.4	105	3477	4134.5	84*
	F	6487	6462.4	100	17885	17931.7	100	1736	1780.8	97	1649	1877.3	88*
Hastings County	M	1878	1955.4	96	6041	5981.6	101	969	771.3	126*	1295	1057.9	122*
	F	1546	1585.0	98	4667	4566.4	102	563	460.1	122*	558	472.7	118
Huron County	M	1105	1392.9	79*	4281	4439.7	96	524	559.8	94	823	639.7	129*
	F	1107	1104.1	100	3638	3671.5	99	364	352.9	103	375	306.7	122
Kenora District	M	856	928.9	92	2415	2710.3	89*	562	351.8	160*	1441	588.5	245*
	F	548	582.0	94	1281	1350.4	95	378	156.4	242*	488	209.4	233*
Kent County	M	2017	2085.9	97	6596	6369.6	104	826	813.5	102	1199	1072.2	112
	F	1600	1691.9	95	5222	4957.1	105	468	491.1	95	477	490.2	97
Lambton County	M	2018	2087.6	97	6467	6184.1	105	737	789.7	93	1339	1182.6	113*
	F	1635	1674.3	98	5055	4690.6	108*	499	477.3	105	590	513.6	115
Lanark County	M	984	1059.5	93	3576	3365.5	106	471	422.5	111	503	482.4	104
	F	851	871.4	98	2976	2786.3	107	325	264.4	123	235	235.3	100
Leeds and Grenville United Counties	M	1591	1745.2	91	5387	5475.5	98	635	686.2	93	925	831.5	111
	F	1339	1452.7	92	4485	4501.7	100	473	430.9	110	397	397.0	100
Lennox and Addington County	M	540	628.6	86	1747	1989.3	88*	304	253.8	120	343	306.4	112
	F	457	462.6	99	1370	1443.1	95	204	142.2	143*	140	136.3	103
Manitoulin District	M	227	287.1	79	937	914.0	103	100	116.5	86	278	127.9	217*
	F	168	193.3	87	611	561.5	109	64	55.6	115	118	54.0	218*
Middlesex County	M	4800	4939.1	97	14425	14897.9	97	2173	1901.1	114*	2645	2735.6	97
	F	4353	4368.1	100	12406	12875.2	96*	1325	1275.8	104	1323	1298.3	102
Muskoka District Municipality	M	716	845.8	85*	2463	2600.4	95	328	326.7	100	498	351.2	142*
	F	616	621.1	99	1760	1803.8	98	178	172.2	103	173	160.9	108
Niagara Region	M	6828	6344.2	108*	18496	18539.7	100	2214	2370.2	93	3149	3481.7	90*
	F	5306	5221.9	102	14122	14117.5	100	1296	1429.8	91	1395	1540.7	91

County	Sex	Malignancies			Diseases of Circulatory System			Diseases of Respiratory System			Accidents, Poisonings and Violence		
		Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR
Nipissing District	M	1271	1269.3	100	4080	3719.8	110*	537	489.6	110	1098	777.4	141*
	F	878	932.1	94	2524	2309.5	109*	282	253.5	111	427	313.7	136*
Northumberland County	M	1046	1202.3	87*	3785	3745.4	101	414	476.4	87	658	548.7	120*
	F	958	973.3	98	3035	3005.0	101	271	289.2	94	278	263.4	106
Ottawa-Carleton Region	M	6709	6611.0	101	20460	19102.1	107*	2570	2465.6	104	3501	4291.3	82*
	F	6743	6565.0	103	18120	17703.0	102	1889	1791.8	105	1876	1999.9	94
Oxford County	M	1475	1729.9	85*	5320	5312.8	100	652	670.6	97	1009	859.5	117*
	F	1377	1437.5	96	4382	4330.4	101	388	420.1	92	396	402.3	98
Parry Sound District	M	718	836.2	86*	2467	2568.6	96	312	324.4	96	563	356.1	158*
	F	510	543.1	94	1482	1525.7	97	226	150.1	151*	227	148.1	153*
Peel Region	M	2409	2506.0	96	6085	6730.9	90*	706	907.6	78*	1721	2026.3	85*
	F	2148	2148.0	100	4727	5058.5	93*	468	574.3	81*	792	823.8	96
Perth County	M	1253	1490.7	84*	4612	4682.5	98	516	586.1	88	785	699.2	112
	F	1230	1251.7	98	3951	4004.0	99	308	381.3	81	350	342.5	102
Peterborough County	M	1711	1739.8	98	5303	5260.2	101	709	673.7	105	975	913.4	107
	F	1445	1517.8	95	4451	4481.5	99	465	443.4	105	414	435.6	95
Prescott and Russell United Counties	M	827	970.9	85*	3084	3042.7	101	452	393.3	115	655	523.0	125*
	F	653	700.2	93	2243	2028.3	111*	263	208.8	126	210	221.1	95
Prince Edward County	M	428	529.0	81*	1666	1659.4	100	247	210.8	117	301	249.2	121
	F	406	432.5	94	1334	1399.8	95	177	136.0	130	137	121.1	113
Rainy River District	M	488	556.6	88	1585	1729.3	92	243	222.2	109	497	295.5	168*
	F	323	351.8	92	955	934.6	102	164	100.2	164*	151	113.2	133
Renfrew County	M	1533	1747.2	88*	5291	5325.3	99	773	698.3	111	1277	986.6	129*
	F	1223	1301.6	94	3806	3668.9	104	463	380.9	122*	445	406.1	110
Simcoe County	M	3088	3517.3	88*	11092	10620.3	104*	1246	1367.3	91	2133	1809.9	118*
	F	2604	2788.1	93	8632	8206.0	105*	728	814.5	89	879	808.8	109

County	Sex	Malignancies			Diseases of Circulatory System			Diseases of Respiratory System			Accidents, Poisonings and Violence		
		Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR
Stormont, Dundas and Glengarry	M	1933	2108.0	92	6508	6508.3	100	909	830.6	109	1291	1073.5	120*
	F	1594	1665.0	96	4956	4990.8	99	616	499.2	123*	484	490.6	99
Sudbury Region and District	M	2505	2370.9	106	7525	6436.9	117*	910	866.5	105	2700	1856.1	145*
	F	1722	1646.1	105	4023	3294.2	122*	395	424.3	93	814	660.4	123*
Thunder Bay District	M	3038	2903.6	105	9443	8480.4	111*	1220	1070.6	114*	2447	1611.9	152*
	F	1824	1892.6	96	5195	4585.2	113*	616	487.7	126*	705	596.5	118*
Temiskaming District	M	991	1028.5	96	3442	3099.1	111*	481	393.7	122*	819	537.9	152*
	F	665	688.7	97	1974	1742.5	113*	223	183.4	122	279	211.4	132*
Victoria County	M	907	1000.3	91	3096	3142.5	99	357	394.0	91	479	401.0	119
	F	753	758.0	99	2408	2366.7	102	253	223.2	113	252	195.8	129
Waterloo Region	M	3461	3855.8	90*	11082	11244.4	99	1108	1459.9	76*	1888	2345.0	81*
	F	3391	3401.6	100	9226	9410.5	98	698	957.3	73*	869	1052.9	83*
Wellington County	M	1883	2120.7	89*	6326	6510.7	97	700	833.7	84*	1049	1103.6	95
	F	1694	1761.9	96	5284	5405.2	98	418	531.3	79*	455	512.9	89
York Region and Metro-Toronto	M	40284	36515.4	110*	102932	105901.0	97*	12958	13370.8	97	16547	21415.8	77*
	F	35074	33642.3	104*	87171	91466.7	95*	8464	9055.2	93*	8997	9774.1	92*

Acknowledgement

This work was begun originally by Dr. R.B. Sutherland, former director of the branch, who assembled much of the data and carried out these analyses for a number of years.

Résumé

On présente les taux de mortalité normalisés à l'échelon des comtés/districts/municipalités régionales en Ontario par périods de cinq années civiles entre 1954 et 1978 pour un certain nombre de causes de décès.

References

[1]Report Relating to the Registration of Births, Deaths and Marriages in the Province of Ontario for the Year Ending 31st December, 1954-1978; Registrar-General of Ontario.

[2]Census of Canada 1956; Dominion Bureau of Statistics.

[3]Census of Canada 1961; Population Age Groups, Dominion Bureau of Statistics. Catalogue 92-542 Vol. 1 Part 2.

[4]Census of Canada 1966; Population Age Groups, Dominion Bureau of Statistics. Catalogue 92-610 Vol. 1; 1968.

[5]Census of Canada 1971; Population Age Groups. Statistics Canada, 1973; catalogue 92-610 Vol. 1 Part 2.

[6]Census of Canada 1976;
Population: Demographic Characteristics Five Year Age Groups. Statistics Canada 1978; catalogue 92-823.

[7]Miettinen OS. Standardization of Risk Ratios. Amer J of Epid 1972; 96, No 6.

[8]Freiman Daniel H, *et al.*
Biometrics 1980; 36, No 2.

Publications Available from the Government Book Store

Publications under the Regulation Act vol. 112-39	
Revised Regulations of Ontario, 1980 Regulation 692/80 Industrial Establishments	$2.00
Revised Regulations of Ontario, 1980 Regulation 691/80 Construction Projects	$2.00
Revised Regulations of Ontario, 1980 Regulation 694/80 Mines and Mining Plants	$2.00
Regulations respecting Lead – made under the Occupational Health and Safety Act Ontario Regulation 536/81	$1.00
Regulation respecting Mercury – made under the Occupational Health and Safety Act Ontario Regulation 141/82	$1.00
Regulation respecting Vinyl Chloride – made under the Occupational Health and Safety Act Ontario Regulation 516/82	$1.00
Regulation respecting Coke Oven Emissions – made under the Occupational Health and Safety Act Ontario Regulation 517/82	$1.00
Regulation respecting Asbestos – made under the Occupational Health and Safety Act Ontario Regulation 570/82	$1.00

Regulation respecting Isocyanates – made under the Occupational Health and Safety Act Revised Statutes of Ontario, 1980 Chapter 321	$1.00
An Occupational Health Nurse's Guide to the Preparation of a Policy and Procedure Manual	$2.00
A Guide to The Occupational Health and Safety Act, 1978	$1.00

News and Notes

The Role of Government Agencies in Radiation Safety in Ontario

Atomic Energy Control Board (AECB)

Radioisotope and Transportation Division (613) 992-7761
Responsible for licensing radioactive materials and for monitoring their use. Call for information on radiation safety related to radioactivity.

Health and Welfare Canada – Radiation Protection Bureau (RPB)

Dosimetry Service (613) 998-3777
Supplies personal monitors ('badges', 'TLDs') for radiation workers.

X-Ray Section (613) 995-9562
Responsible for administering the parts of the *Radiation Emitting Devices (RED) Act* and regulation, which apply to manufacturers and suppliers of new x-ray equipment.

Non-Ionizing Radiation Section (613) 995-9801
Administers the parts of the *RED Act* applying to manufacturers and suppliers of equipment producing non-ionizing radiation, such as radiofrequency, microwave, visible light, ultraviolet and infrared.

Ontario Ministry of Labour

Radiation Protection Service (416) 965-8178
Responsible for registration, plan approval and inspection of all non-medical x-ray installations. Also responsible for occupational radiation safety in all Ontario workplaces other than those that are owned by the federal government, and including both x-rays and non-ionizing radiation.

Ontario Ministry of Health

X-Ray Inspection Service (416) 963-1035
Responsible for registration, plan approval and inspection of all medical and dental installations. Call for information on patient or public exposures to medical and dental x-rays.

Director of Canadian Centre for Toxicology Appointed

Ian C. Munro, formerly Director General, Food Directorate, Health Protection Branch, Health and Welfare Canada, has been appointed director of the Canadian Centre for Toxicology.

The new centre, which is jointly sponsored by the federal and provincial governments, industry and the Universities of Toronto and Guelph, will provide a focus for research, teaching and testing services to meet the developing needs in this field.

The centre will have facilities on the campuses of the Universities of both Toronto and Guelph, and construction is expected to be completed within the next four years.

Dr. Munro, who is a graduate of McGill University and who obtained his doctoral degree in toxicology and pharmacology from Queen's University, is currently chairman of the Tripartite Toxicology Committee (U.S., U.K. and Canada), and is a member of the International Life Science Institute Experts in Pathology and Toxicology.

He has, in the past several years, been active on 25 major national and international task forces and committees related to food safety or health, and is past chairman of the International Committee on Hazards Associated with Dioxin in the Great Lakes.

As director general of the Food Directorate, Health and Welfare Canada he was responsible for his branch's activities in the areas of microbial hazards, nutritional sciences, chemical safety and other matters related to health and safety.

Throughout his career Dr. Munro has continued an active involvement in research in conjunction with his administrative duties and has written more than 60 scientific papers.

Occupational Health and Safety Chair at University College of Cape Breton

A Chair of Occupational Health and Safety has been established at the University College of Cape Breton. Sponsored by the Atlantic Foundation for Occupational Health, the Chair will serve as a focus for the educational and practical research and development programs that promote the health of people in the workplace.

Dr. Albert Prossin has been appointed University Professor and he has consented to be the first to hold the Chair. Dr. Prossin, internationally known in occupational health and safety, will continue as president of the Atlantic Foundation.

The University College of Cape Breton has been a leader in the development of occupational health and

safety education in Atlantic Canada over the past eight years. This major announcement marks another significant step in the practical application of university programs to community needs.

The establishment of the Atlantic Foundation Chair of Occupational Health is a milestone in the continuous effort to build a comprehensive service for workers and employers in both on-shore and off-shore work sites. This reflects the considerable support given by the Government of Nova Scotia and the Cape Breton Development Corporation to the Atlantic Foundation for advances in the health and safety of workers.

Dr. Prossin will bring community resources, as well as the co-operation of all the Atlantic Foundation, to his new post at UCCB. This major development at the university marks another significant advance in making the university resources and programs available to the real needs of the people in Atlantic Canada.

Three-day intensive course in hygiene theory and practice offered by the Occupational and Environmental Health Unit, University of Toronto, and specially designed for those newly entering the field of hygiene.

February 15-17th, 1984, Toronto Canada

The course will emphasize risk assessment in the workplace, using quantitative methods. Topics will be presented in lecture sessions followed by practical laboratory exercises to give participants both theory and hands-on experience. The topics will include: Air sampling pumps with calibration and basic maintenance, solid sorbent sampling, filtration methods, liquid absorption, direct reading instrumentation and noise and heat stress evaluation. The development of sampling strategies and control methodologies will be discussed.

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Coming Events

February 22, 1984	Occupational Hygiene Association of Ontario 'Reproductive Toxicity Symposium and General Meeting'. Toronto.
May 7-9, 1984	International Conference on Occupational Ergonomics. Harbourcastle Hilton Hotel, TORONTO, CANADA. Details from R.D.G. Webb, Technical Chairman, Toronto '84, P.O. Box 1085, Station 'B'., Rexdale, Ontario, Canada M9V 2B3. Tel. (416) 675-2235.
May 22-25, 1984	ARBO 84 – International Trade Fair for Safety, Health and Welfare at Work – RAI Amsterdam. Details from RAI Gebouw bv, Europaplein, 1078 GZ, Amsterdam.
July 10-12, 1984	Society of Occupational Medicine Annual Provincial Meeting. York, England.
August 20-24, 1984	Third International Conference on Indoor Air Quality and Climate – Stockholm, Sweden. Details from Mr. P.R. Warren, Building Research Station, Garston, Watford, WD2 7JR. Tel. 09273 74040.
September 9-14, 1984	21st International Congress on Occupational Health, Dublin, Ireland. Details from Dr. C.S. Macnamara, Organizing Secretary, XXI International Congress on Occupational Health, 44 Northumberland Road, Dublin 4, Ireland.
September 24-26, 1984	4th International Congress on Industrial and Environmental Neurology. Czechoslovak Medical Society, J.E. Purkyne, Vitëzneho února 31, 120 26 PRHA 2, Czechoslovakia.
October 29-November 2, 1984	Ontario Occupational Health Nurses' Association Annual Conference, Windsor, Ontario. Co-hosts: Mary Weston and Barbara Brew, % G.M. Trim Plant, 1600 Lauzon Road, Windsor, Ontario N9A 6R9. Tel. (519) 944-8226.
December 3-5, 1984	Inter-Noise 84 – 13th International Conference on Noise Control Engineering – Honolulu. Details from the Inter-Noise 84 Secretariat, P.O. Box 3469, Arlington Branch, Poughkeepsie, NY 12603, USA.

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Ministry of
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